Webber (S. G.) 90

THE

PATHOLOGICAL HISTOLOGY

OF THE

SPINAL CORD,

WITH

A SHORT INTRODUCTION ON THE NORMAL HISTOLOGY.

BY

S. G. WEBBER, M.D.,

VISITING PHYSICIAN FOR DEPARTMENT OF NERVOUS DISEASES, CITY HOSPITAL.

Reprint from Medical and Surgical Reports of City Hospital, Third Series.

BOSTON:
PRESS OF ROCKWELL AND CHURCHILL, No. 39 ARCH STREET.
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THE PATHOLOGICAL HISTOLOGY OF THE SPINAL CORD.

By S. G. Webber, M.D.

Many of the specimens from which sections have been made for study in preparing this paper came from hospital patients. The design of the paper, and the space at the author's disposal, have made it necessary to exclude all clinical histories. It has also been necessary to refer only very briefly to the investigations of others, and to omit many discussions which would be very interesting. The author has endeavored to give proper credit to others in quoting their views.

The usual method of preparing clear sections of the spinal cord has been employed after the cord was hardened in chromic acid or bichromate of potassa. The drawings have all been made with the camera lucida. The microscope used was made by Hartnack; objective No. 8 was used for nearly all the drawings, except as mentioned below.

NORMAL HISTOLOGY.

The grosser divisions of the cord need not be dwelt upon, nor is it necessary to describe its envelopes, excepting to say that the pia mater, next to the cord and surrounding it, sends processes into its interior, accompanying the blood-vessels distributed to the different parts of the organ. Between the pia mater and the nervous structures of the cord is a layer of connective tissue, thickest where the nerve-roots pass out and at the posterior fissure. This cortical layer is formed of fine fibres, woven into a net-work with extremely fine meshes. Scattered among these fibres are cellular elements. The thickness of this cortical layer is from .01 to .06 millimetre. (Frommann.) (Fig 1, *a*.)

The cortical layer sends processes into the cord accompanying the pia mater, and also others, forming septa (Fig. 1, *b*, *b*), which divide and subdivide, finally forming a net-work of fibrous tissue, which surrounds and supports the nerve fibres, taking the place of the sheath of Schwann. (Bidder, Schultze, Frommann.)

The structure of the connective tissue, neuroglia (Virchow), is one of the most important studies in the normal histology of the cord, in order to obtain a correct basis for the study of pathological processes. It is only within comparatively recent years that the presence of connective tissue in the cord has been generally recognized. (Bidder und Kupffer.) Frommann describes the neuroglia as consisting of a very thick net-work, with narrow meshes, formed in part of interlacing fibres, and in part of cells with numerous processes.

There has been considerable difference of opinion in regard to the structure of the interstitial tissue. Later observers are inclined to the view that this tissue is composed of fibres and cellular elements. My examinations tend to show that this is the correct view.

The fibres of the neuroglia in the white substance of the cord form a net-work in the larger septa nearly as close and fine as in the cortical layer; but as these trabeculæ subdivide, the fibres finally are interlaced around the nerve fibres, and on cross-section the meshes appear more open. The general direction of these fibres is nearly parallel with the nerve fibres; but there is great irregularity, and they cross each other and the nerve fibres at all angles.

CELLULAR ELEMENTS OF THE NEUROGLIA.

NUCLEI.

Nuclei are found scattered through the cortex and the trabeculæ. These nuclei are round or oval; they may have no distinct nucleolus, but are filled with granules, two or three of which are more prominent than the others, and have more or less lustre. The outline of

these nuclei is well defined. They have received the name of myelocytes. (Ch. Robin.) A careful study of these nuclei leads to the conclusion that they are surrounded by protoplasm, apparently without cell-wall.

DEITERS' CELLS.

Besides nuclei, there are found cells scattered throughout the interstitial tissue, which have been named from Deiters, who first described them as a mass of fibres radiating from the nucleus in which there is no nucleolus. These fibres have a firm but delicate appearance, and divide frequently.

Franz Boll (Arch. f. Psychiat. u. Nervenkr., IV., 1873, p. 1) describes these cells, and ascribes to them more importance in the structure of the spinal cord than others. "I recognize in the white substance of the spinal cord no separate connective tissue fibrillæ, and no corpuscles without processes, but only the above-mentioned cell described by Deiters, with its numerous fibrilliary processes." Attached to the fibrillæ were granules, probably of albuminoid nature. He has never seen the processes divide.

C. Lange describes these cells as consisting of a rather small and thin cell-body, often long in form; a nucleus also long. The body of the cell is drawn out into processes, on an average at least seven, separated from each other by relatively deep notches. He says it seems as if these cells and their processes together form the interstitial tissue.

It is necessary to add but little to these descriptions. I have frequently seen nucleoli in these cells; and in many instances, with a high power, have seen two or three fine fibres radiating from the nucleolus. (Fig. 4, *c*.) I have not been able to follow these beyond the nucleus. In most cases, however, only the nucleus is visible, filled with coarse granules, generally situated near one end of the rather long cell.

The number and distribution of these cells is far from uniform. They also vary in size, in the number of processes they give off, and the manner in which they are

grouped together. Sometimes several will be found near each other apparently connected. (Fig. 2.) On longitudinal section, the cells, as well as nuclei, are frequently seen in groups. These cells are found scattered irregularly among the nerve fibres generally, not in the trabeculæ which enter the cord from the cortical layer. The processes form meshes which vary in size according to the size of the nerve fibres which they surround. In the inter-spaces between the fibres are seen, on transverse sections, the ends of fibres running a course more or less longitudinal. With a low power these cut ends present a granular appearance.

Frommann (Untersuch. ü. die norm. u. patholog. Anat. des Rückenmarks, 1. Theil, p. 46) considers the fibres, both coarser and finer, to be cell processes. He believes they are hollow tubes or canals, and that, with the cells, they form a vast canal system for the circulation of the fluids in the spinal cord.[1]

The interstitial tissue is formed of fibres closely woven together, crossing each other in all directions, with cells and nuclei intermingled. Possibly a certain amount of serous fluid contained in this tissue may coagulate on the application of reagents. Granules arising from the coagulation of this fluid may adhere to the fibres, and at times give them a roughened appearance.

NERVE FIBRES.

The axis cylinders, and medullary sheath of the nerve fibres in the spinal cord require no special description, resembling very closely those of the peripheral nerves.

The size of the fibres varies according to the region of the cord under examination, the smallest being in the central posterior columns, where the size is quite uniform. The largest fibres are found along the anterior fissure and the anterior portion of the circumference. In the lateral columns there is the greatest variation in size.

[1] An exhaustive account of the neuroglia may be found in Frommann's work above cited, with details into which it does not seem necessary to enter.

As already mentioned, opinions differ in regard to whether the nerve fibres have a distinct sheath. In fresh healthy specimens I have been unable to discover any such sheath. The fibres have seemed to be closely and immediately enveloped by the neuroglia.

GRAY SUBSTANCE.

The gray substance is composed of all the elements heretofore described, and also of nerve cells. The nerve fibres and the fibres of the neuroglia are irregularly distributed and intermingled. Many of the nerve fibres are reduced to their axis cylinders, and are sometimes so fine as to be distinguished with difficulty from the fibres of the neuroglia.

NERVE CELLS.

The nerve cell consists of a cell body, with one to several processes, containing a nucleus more coarsely granular than the cell body, and which is clearly defined, round or oval; the nucleus contains a finely granular or homogeneous nucleolus, in which may appear a light-colored spot, a sub-nucleolus. At one end of the cell there are frequently coarse yellow granules, especially if the person is aged.

Under fortunate circumstances, from one to three or four fine fibres or lines may be seen arising from the nucleolus or sub-nucleolus, which run a short distance and are lost in the body of the cell. (Figs. 5, 6.) Frommann has described these fibres at length. (Virch. Arch., XXXIII., 1865, p. 168.)

The body of the cell, and, to a certainextent, the processes, are finely fibrillated. These and other minute markings probably change quickly after death.

One of the processes, a short distance from the cell, becomes clothed with a medullary sheath, and takes the structure of a fully developed nerve fibre; the others subdivide into extremely fine fibres, and assist in forming the fine net-work of the gray substance. Opinions differ as to whether these fibres belong to the proper nervous elements. This description applies to the stellate cells; fusiform cells send a process from each end, which can be followed for a long distance without division.

The largest cells with most processes are found in the anterior cornua, and along the external border of the gelatinous substance in the posterior cornua. Fusiform cells, both large and small, are found in the posterior cornua, are almost the only cells in the *tractus intermedio lateralis*, and are also sparsely scattered in the anterior cornua. The smallest cells in the cord are in the posterior cornua, and are fusiform in shape. (Figs. 9, 10.)[1]

COMMISSURES AND CENTRAL CANAL.

The lateral halves of the cord are connected by the anterior or white commissure, and the posterior or gray commissure, respectively anterior and posterior to the central canal, which runs through the whole length of the cord, opening above into the fourth ventricle, below into the posterior fissure. The central canal is clothed with cylindrical epithelium. The epithelium consists of long cells with large nuclei; their free ends have ciliæ projecting into the central canal; from the opposite end proceed fibres which can, in a favorable specimen, be followed into or across the commissures. (Fig. 11.)

Around the central canal is a narrow zone of translucent tissue, *substantia gelatinosa centralis*. (Stilling.) Its translucency is due to the almost entire absence of nerve fibres. The nuclei scattered through this zone may be so multiplied as to obscure the translucency. These nuclei may also encroach upon the central canal, causing it to disappear. Sometimes the epithelium lining the canal increases so as to fill and obliterate it.

VESSELS.

The vessels of the spinal cord do not require special description. It has, however, been a disputed question whether they are surrounded by a perivascular space like the cerebral vessels. His described such a space with a lining membrane or sheath. Leyden has accepted this as a fact. Fig. 12 shows a small vessel, almost a capillary, with a perivascular sheath containing only a few nuclei; the vessel has collapsed.

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[1] J. L. Clark has given a very full and detailed description of the distribution of the different kinds of nerve cells. — Philos. Trans., 149, 1859, p. 437.

PATHOLOGY.

NEUROGLIA.

NUCLEI.

One of the earliest changes in the interstitial tissue is an increase in the number, and, perhaps, in the size, of the nuclei and cells; there is also a corresponding increase of the fibrous structure. The nerve fibres in the white substance are pressed apart and do not lie so near each other as in the normal state. (Fig. 13.) Kesteven (St. Barthol. Hosp. Rep., 1872, Vol. VIII.) says this increase of nuclei is probably the starting-point of sclerosis. C. Lange (Schmidt's Jahrb., Bd. 168, 1875, p. 238) describes the increase of free nuclei scattered through the fibres of the reticulum in myelitis ending in softening. They may be grouped together in large numbers, may give rise to a granular appearance, and, he says, are adherent to the fibres.

Most authors do not distinguish between the more advanced and the earlier stages of the degeneration. In the former the nuclei are often increased in number, enlarged in size, and grouped in clusters of two or three to eight or ten. These groups are less common in the earlier stages of the disease. The nuclei are sometimes smaller than normal. In shape they are round or oval, — the long axis only slightly longer than the short axis; when there is a great difference, the nuclei probably belong to blood-vessels.

In the gray substance it is less easy to recognize a slight increase in the number of nuclei, their distribution being less regular and their numbers more numerous, normally, than in the white substance. In advanced stages of disease the increase of nuclei in the gray substance is very evident. (Hayem, Pierret, Charcot and Joffroy, Roger and Damaschino.) Damaschino says they are remarkable for their homogeneous appearance: that they enclose two or three punctiform nucleoli, and show no tendency to fatty degeneration; they are most abundant around altered blood-vessels, adhere to their adventitia, and obscure the observance of their structure. (Mem. de la Soc. de Biol., 1871, p. 49.)

They are also scattered through the whole extent of the diseased tissue without special reference to the blood-vessels. In the gray substance they are smaller, as a general rule, than in the white.

When portions of the hardened diseased cord are teased apart, these nuclei are, in many cases, seen surrounded by small portions of finely granular substance. (Fig. 14, *a*.) It is difficult to decide whether this is a portion of the diseased tissue mechanically adherent or protoplasm: no cell-wall can be seen bounding this adherent granular substance. Charcot mentions that the nuclei are thus surrounded by protoplasm. (Arch. de Physiol., 1871, t. IV., p. 93.)

FIBROUS TISSUE.

The increase of the fibrous tissue of the white structure is easily recognized, as the trabeculæ and meshes enclosing the nerve fibres are thickened. In chronic diseases the fibres are much finer than normal, and are closely interwoven, having an appearance suggestive of felt. The cut ends of the fibres, showing mere points, give a granular appearance to sections of hardened cord. On teasing apart these portions, it is found that the diseased parts are composed of fibres, and not of a granular substance. Where the disease is far advanced this increase of fibres is yet more marked: on transverse section the whole field has a granular appearance, only a small number of fibres are seen crossing the field, sometimes in bundles, sometimes separate: nuclei are scattered over the field. On longitudinal section the fine fibres are seen running parallel with the long axis of the cord, often with a wavy appearance, well described by Frommann. He gives a detailed account of this new-formed fibrillary tissue which is in general very accurate: but some of the appearances I have not found as he describes. (Untersuch. ü. die norm. u. pathalog. Anatomie des Rückenmarks, 2. Theil, 1867, p. 88.) He says that at the border where the change is least advanced, the fibres are thickened, being two or three times the normal thickness, or are bordered by a dully shining, sharply defined, translucent substance. From the edge of many fibres arose small excrescences or processes. A

similar appearance is described in the more advanced stages of the disease: he also mentions individual, free fibrillæ running more or less transversely; he also speaks of fibres containing fibrillæ, and of a granular "foundation substance."

I have been able to find only fibres, nuclei, and cells. The foundation substance, or membrane, is represented, at most, only by a fluid which bathes the fibres, and is coagulated by the reagents used in hardening. The fibres have been uniformly smooth, without the knotted processes. When separated one from another they were no larger than in the normal condition, perhaps were rather thinner, and nothing resembling fibres composed of fibrillæ were seen (excepting as shown in Fig. 21, which will be described later). The semi-translucent border which he describes was not seen. These fibres generally run parallel with the nerve fibres, or the remaining axis cylinders, and have the wavy appearance described by Frommann. Comparatively few run transversely. This longitudinal course of the fibres fully explains the granular appearance of transverse sections.

In *acute* degeneration, the neuroglia fibres have been described as swollen, thickened, and twice, or more than twice, as large as normal. These swollen fibres, three or four times the normal size, had lost their shining appearance, had taken a finely granular character, and were strongly colored by carmine; in many places neighboring fibres had run together so that there were patches where was a continuous connective tissue substance. (Frommann, Hayem.)

CELLS.

The neuroglia cells are increased in size and number. These cells sometimes seem to consist merely of an increase of protoplasm around the nuclei without clearly defined cell-wall, and without processes. These simple forms of cells can be seen much the more readily in longitudinal sections.

DEITERS' CELLS.

Previous to the description of these cells as normal constituents of the cord, Rindfleisch had seen and described them as one of the products of inflammation. (Virch.

Arch., XXVI., 1863, p. 474.) They have been described by Charcot, Jeffroy, Hallopeau, Gombault (with Charcot), Schüle, Maier, Lange, Frommann, and others. These descriptions do not agree in all respects; but the differences can be explained by the fact that the cells have been seen in different stages of change, or after different methods of treatment.

Joffroy (Mem. de la Soc. de Biol., 1873, p. 77), in myelitis produced artificially in a dog, found a great number of long and very fine fibres, not ramified, starting from a centre represented by a very limited mass of protoplasm, which was very strongly colored by carmine; these contained neither nucleus nor nucleodus. He considered these to be Deiters' cells, and that they play an important part in the evolution of inflammation.

Maier (Virch. Arch., LXI., p. 1) speaks of large, star-shaped bodies, with many processes, having an amorphous, granular contents, the cell body frequently including other cell structures. He says these are the hypertrophied cells of the neuroglia.

These spider cells are enlarged and increased in numbers in both acute and chronic inflammation, in both the white and the gray substance. They may contain a single nucleus, generally eccentric, almost always oval, generally finely granular, sometimes with two or more granules larger than the others; rarely there is a distinct nucleolus to be seen. From this cell arise processes radiating in all directions; sometimes the cells are homogeneous and show no structure, in which case they imbibe carmine so as to present a smooth, red, amorphous appearance, with no markings nor lines excepting the nucleus. The outline of the cell is irregular, the cell-wall falling in towards the nucleus between the processes, and sometimes the cell spreading out into broad tracts of protoplasm, from which arise several processes. These processes vary in number and delicacy, and are so fine and numerous as to be counted with difficulty (Fig. 15, *a*); then again they are finer and coarser; occasionally, though rarely, they subdivide (Fig. 20); some can be followed only a short distance, others for a long distance. Sometimes these cells

are fusiform in shape, with the processes grouped at the ends (Fig. 16); these closely resemble cells described by Boll, found in the brain and spinal cord of oxen, sheep, etc.

Occasionally these cells and their processes are much swollen and are irregular in their course, being twisted and doubled upon themselves so that it is impossible to follow them far: the nucleus is much obscured, and is sometimes invisible. (Fig. 17.) Such cells imbibe carmine readily and acquire a deep red color.

In a case of sclerosis, cells were seen with two or more nuclei. These cells were relatively large, and were found at the boundary of the diseased tissue. Many cells contained two nuclei, showing perhaps the commencement of division. (Fig. 15, *a*.) At (Fig. 18) is drawn a cell with five round nuclei closely grouped together; a careful examination showed these to be all in the cell. The cells had a perfectly homogeneous appearance without markings nor granules; from some of them arose processes wider than are usually seen, having the same homogeneous structure. These cells resembled more closely than others the appearance which might be caused by a liquid forcing itself into the interstices of the neuroglia, and spreading out into the free or cellular spaces around the nuclei. Some authors indeed consider these cells to be channels for the nutritive fluids, or lymph; hence the name "Saftzellen."

In other instances these cells seem to have undergone atrophy, the cell body being only slightly larger than the nucleus, only a few processes remaining. (Fig. 19, *a*.) These are found where the disease is far advanced.

Owing either to differences in structure or changes during the hardening it is often not possible to see the nucleus in these cells, there is only an appearance of fibres crossing each other, or taking their origin from a confused knotted centre: hence the name knotted points (Knotenpunkten), given by some authors.

Joffroy (Mem. de la Soc. de Biol., 1873, p. 77) says that Charcot has found these cells in every case of sclerosis and ataxia where he has sought them.

In sclerosis these spider cells are often united by one or two of their processes with the sheaths of the blood-vessels. (Fig. 20, *s*, 24, *c'*.) This connection of the cells with blood-vessels would confirm the view that they are lymph canals. (Arndt, Frommann, Leyden, Meynert.) Arndt gives as the reason for believing them to belong to the lymph system that they undergo hyaloid degeneration when the vessels are thus affected, and when the perivascular spaces are overfilled with fluid they swell. (Arch. f. Psych. u. Nervenkr., II., p. 755.)

One cell has been seen with two processes which split up into fine fibrillæ, which were lost in the surrounding neuroglia. (Fig. 21, *a*, *a*.)

Charcot and Gombault (Arch. de Physiol., t. v., 1873, pp. 143, 304) have described a case of syphilitic disease of the brain and spinal cord, in which these spider cells play an important part. The transformations through which they may pass are described. The nucleus may assume a granular appearance and lose its distinctive character, becoming a mere mass of granules: the cell may undergo fatty transformation, may lose its processes, and finally be represented by a round mass of granules; it has entirely lost its characteristic structure and may disappear. Another change is less clearly explained: "Two hypotheses seem to us admissible; either the tissue has passed through the caseous period and has undergone repair after absorption of the degenerative elements, or the irritation was from the beginning less severe, the morbid process went on more slowly, and the tissue was organized progressively without undergoing fatty degeneration in any part."

The above descriptions apply to the changes which these cells undergo when the disease, affecting chiefly the neuroglia, is of a chronic nature, as in sclerosis. Leyden describes the same increase in size and number as occurring in acute myelitis, but considers that these cells belong more especially to the third stage, that of resorption; he refers their presence and their large size to contraction of the tissues, arising from resorption of degenerated elements causing a dilatation of the nutritive spaces.

Joffroy, Charcot, Hayem, have found these cells in cases of acute myelitis.

A large proportion of the new fibrous tissue in chronic myelitis has its origin from these cells, and it is not unreasonable to suppose, with many authors, that all the newly formed fibrous tissue thus arises from these cells.

Where the nerve structures are chiefly affected, there being little or no increase of the neuroglia, the spider cells do not suffer material change: they have their distinct characteristics, are not increased in number or in size.

OTHER CELLS.

Frommann (Virch. Arch., Bd. 54, p. 42) describes cells which had a finely granular protoplasm, generally with one or two nuclei; these cells filled or partly filled the spaces formerly occupied by nerve fibres. They were occasionally found with the axis cylinders still present, and were then either to one side of the axis or surrounded it. He expresses no opinion as to the nature of these cells.

Leyden (Arch. f. Psych. u. Nervenkr., VI., p. 278) describes, in a case of infantile paralysis, large, pale, round cells, with rather sharp boundaries, clear contents, and clearly defined nuclei. He speaks of them as endothelial structures, their significance not being clear, but thinks that they arise from the neuroglia, by swelling and division of elements. He compares them to the granular cells. He found them also in rents in the cord, in a case of exposure to diminished atmospheric pressure. (Ibid., IX., 1879, p. 316.)

These cells, as he describes them, resemble very closely the nuclei surrounded with protoplasm already described. They differ in that Leyden's cells are said to have a sharp contour. In the gray substance the nuclei are abundant, and it would be expected that they should increase in number in disease.

Kahler and Pick (Arch. f. Psych. u. Nervenkr., X., 1879, p. 186) speak of these cells as occurring in myelitis from compression (fracture). They consider them endothelial cells, arising from the endothelium of the lymph spaces. They give no drawings. Their descriptions are very similar to Leyden's.

NERVE FIBRES.

Both the axis cylinder and the medullary sheath may be affected, or either alone. The earliest change in acute myelitis is swelling. The medullary sheath acquires a diameter three or four times the usual size, and the concentric markings disappear. In the fresh state the medullary sheath is replaced with fatty, granular bodies, which are dissolved in the process of clearing up the section; but there remains frequently a faint appearance of coagulated debris.

Occasionally a perfectly homogeneous, structureless sheath is seen with the axis cylinder in the centre. (Fig. 13.) With polarized light, healthy nerve fibres, on transverse section, show a marking resembling that seen on some kinds of starch. The axis cylinder is dark, and forms the centre of a Greek cross, the quadrants between the arms of the cross appearing bright. Nerve fibres which are enlarged, and have undergone a change in the structure of their myeline, either present no such appearance, or it is irregular and imperfect. This examination is best made in specimens cleared up with oil of cloves, etc.

In cases where the neuroglia is increased, interstitial myelitis, sclerosis, the nerve fibres finally entirely disappear; this occurs, however, only in extreme cases; generally many of the axis cylinders persist; the axis is then closely encircled by the fibrous tissue, is often of less size than normal, and the medullary sheath has completely disappeared (Fig. 13, *a*), having been either absorbed without special change, or more probably having undergone fatty degeneration previous to absorption.

The axis cylinder may be greatly enlarged, sometimes to ten or twelve times the normal size. (Figs. 27, 28.) This is most frequent in acute myelitis. This hypertrophy does not extend over a long tract without interruption; the enlargement is globular or fusiform in shape. The axis cylinder may undergo several such enlargements within a short distance, which gives rise to a varicose appearance. The enlarged axis cylinders, if near together, are so affected as to fit into and overlap each other. (Fig. 28.) Occasionally small vacuoles

are to be seen in the enlarged axis cylinder (Fig. 28, *a'*) : the significance of this is not known, nor their mode of formation. Many of the axis cylinders have a decided granular appearance : sometimes this is most marked around the circumference, while the centre is darker, giving an appearance resembling a nucleus.

The myeline is not increased in amount : at first it forms a thin ring around the axis cylinder ; it undergoes change which ends in its absorption and disappearance. This destruction of the myeline does not always keep pace with that of the axis cylinder ; the latter may entirely disappear, while coagulated masses representing the myeline still remain.

The enlargement of the axis cylinder is found most clearly marked around the periphery of spots whose centres have undergone more extensive changes ; the neuroglia is comparatively unaffected. The final result is the entire destruction of the nervous elements.

The hypertrophy of the axis cylinders was seen seventeen hours after the beginning of the disease, by M. Roth (Virch. Arch., IV., 55, 1872, p. 197), and twenty-four hours after division of the cord by Charcot. (Arch. de Physiol., IV., 1871, p. 93.)

Roth rightly thinks this change to be an active process, an hypertrophy, not merely a secondary change. Joffroy (Mem. de la Soc. de Biol., 1873, p. 77) considers that it partakes of the nature of an irritative process, a parenchymatous inflammation. He says it is never found with fatty degeneration of the nerve fibres ; hence he concludes that tumefaction of the axis cylinders and fatty transformation of the myeline belong to two different processes. This tumefaction, he says, plays no part in ascending or descending secondary changes. Other authors find enlarged axis cylinders in chronic myelitis, as in sclerosis ; but then the change is less extreme. The axis cylinders in both the white and gray substances may be affected.

Softening and final disintegration come from the fatty degeneration and the breaking up of all the tissues. Lockhart Clarke (Med. Chir. Trans., XLVIII., 1865, p. 264) describes the different stages of softening at length, but Leyden (Klinik,

Bd. II., p. 132) gives a much better description of the process. He says, in substance, that the nerve fibres in certain spots have a swollen appearance; by the intrusion of numerous and enlarged granular cells they are pressed apart, are irregularly compressed, are diminished in size, and deprived of their myeline. The medullary sheath becomes dark, muddy, and finally fatty. Fat drops are also scattered about between the nerve fibres. The ganglion cells suffer disintegration and atrophy. The firmness of the connective tissue is decidedly diminished, and there remains a crumbling, brittle, fragmentary mass. The same process may be observed in the gray substance, allowance being made for differences in the normal structure.

Charcot (Arch. de Physiol., III., 1870, p. 247) reports a case of glosso-laryngeal paralysis with atrophy and disappearance of nerve cells in the anterior cornua, but with no diminution in the number of nerve fibres nor increase of nuclei. It is quite unlikely that the nerve cells should be thus affected and the nerve fibres entirely escape: it is more probable that so few nerve fibres were affected that the change was overlooked.

Unless the nerve elements are primarily affected the axis cylinder persists, almost indefinitely; even where the increase of connective tissue is extreme in the white substance, and where the surrounding gray substance is almost ready to break down, the axis cylinders may still be seen. When the nervous elements are chiefly affected, in parenchymatous myelitis, the axis cylinders quickly disappear.

NERVE CELLS.

The nerve cells of the anterior cornua have been most thoroughly studied, and their pathological changes have been most frequently described: those of the posterior cornua have attracted much less attention, indeed, have generally been entirely overlooked.

In chronic diseases the earliest, or one of the earliest, changes in the nerve cell is an increase in the amount of deep yellow pigment: the processes are shortened and soon entirely disappear: at the same time with these changes the

cell diminishes in volume, loses its irregular outline, and becomes globular. The nucleus and nucleolus at first retain their normal size and appearance (Fig. 22, *c*, *d*), or the nucleus may become angular (*a*) and the nucleolus be invisible; finally both nucleus and nucleolus entirely disappear, and there remains a small mass of yellowish granules (*e*, *f*), which are in the end absorbed. The diseased cells may be seen scattered among other healthy cells, or all the cells of one group may be affected; or, sometimes, all those of one anterior cornu.

It must be remembered that the cells vary much in shape and size in different regions, and even in the same region; yet those in the same region closely resemble each other. In judging as to disease of the cells this must be considered; thus, in the anterior cornua, a number of globular cells, with only one or two processes, would show disease; but in Clark's columns this is the normal character of the cells; also in the anterior cornua a fusiform cell with only two processes is seen but rarely, and many of these, especially if small, would show disease; but in the *tractus intermedio lateralis*, and in the posterior columns, this is the more frequent shape, and the prevailing size is small.

In proportion to the atrophy of the nerve cells the nerve fibres also suffer, and when a large number of cells have disappeared there is an evident decrease in the number of nerve fibres.

Sometimes the cells are described as uniformly diminished in size, without other change. It is not likely that this is pathological; the cells vary much in the same person, and especially in different persons, and if there is no change in pigmentation, in processes, nor in nucleus, it is not right to infer disease merely from apparent diminution in size.

Sometimes an increase of interstitial tissue supplies the loss of substance occasioned by destruction of nerve elements; but frequently this loss is not made good, and then the gray substance is distorted in shape. Frequently the cells and nerve fibres are alone affected. The disease must be considered in such cases as primary. Gombault reports such a case. (Arch. de Physiol., 1873, p. 80.)

Charcot and Gombault (Arch. de Physiol., 1875, p. 735) mention a case where the cells of the anterior cornua were atrophied while the cornua retained nearly their normal dimensions because of the "truly enormous development which the capillary circulation of that region has received."

In those instances where the cells seem chiefly affected Charcot thinks that they are the primary seat of the lesion.

In acute myelitis the nerve cells are first swollen: they are also found swollen in some cases not acute when the surrounding tissue is also affected. Pierret (Arch. de Phys., 1874, p. 968) found swollen cells in animals with myelitis produced experimentally. The nucleus may be excentric, quite near the periphery.

Fatty degeneration is occasonally found, less frequently, perhaps, than in the nerve cells of the brain. (See Magnan, Compt. Rend de la Soc. de Biol., 1869, p. 113.)

Jolly, quoted by Leyden, and Leyden (Klinik. Bd. 1) regard nerve cells with two nuclei as pathological, but no other author mentions this.

A vitreous change of the ganglion cells is found with partial loss of their processes: in some the vitreous change invades the processes also. These cells are normal in size, or they may be hypertrophied; they have little or no pigment, and sometimes contain cavities. (Hayem, Frommann, Aufrecht, Leyden.)

Cavities, or vacuoles as they have been called, are found within cells either singly or in groups. Kahler and Pick have seen twenty-five in one cell. The septa may break down whereby a large cavity is formed from two or more smaller cavities. Edes (Boston Med. and Surg. Jour., July 24, 1879) reports a case where the cavities encroached upon the processes and caused an enlargement of the cell. Dejerine (Arch. de Phys., 1876) found vacuoles in a case of muscular atrophy and paraplegia.

COMMISSURES AND CENTRAL CANAL.

Changes found in and around the central canal are considered important by some authors. They may indeed give rise to symptoms, but there are usually other changes, and it

is impossible to decide to which the symptoms are due. (Hallopeau, Michaud, A. Eickholt.)

The central canal varies much in size even in health. It may be greatly dilated, as in cerebro-spinal meningitis and in some cases of myelitis and sclerosis. In myelitis the canal is the more frequently obliterated by an increase of nuclei or cells which gradually crowd the epithelial lining into the canal; then the epithelial cells give place to merely nuclear elements, or to very small cells with large nuclei and scarcely any protoplasm. The nuclei may extend laterally and antero-posteriorly, so as almost completely to destroy the anterior and posterior commissures. As the large vessels on either side of the central canal are more or less implicated in this mass of new-formed nuclei the nutrition of the cord in such cases is probably disturbed.

When the canal is not obliterated it may be distorted in shape, and become angular or divided, so as to form a double canal, from the irregular growth of the nuclei. The canal may be filled with a fluid which hardens under the influence of reagents and takes a deep red color from carmine. A similar exudation is sometimes found in other parts of the cord in cases of myelitis. Some authors call this exudation colloid. (Hayem.)

Changes in the commissures are independent of the condition of the neighboring white substance. Where the nerve fibres were destroyed close up to the commissures there was in some sections no material increase of nuclei, only a moderate multiplication of the epithelium. In another section there were also fibres crossing between the epithelial cells in an obliquely antero-posterior direction. A similar change is described by Hallopeau (Mem. de la Soc. de Biol., 1869, p. 168) in a case of *sclerose diffuse périépendimaire*.

The relation which an increase of nuclei in the commissures bears to morbid processes is not yet well determined. In myelitis, with extensive destruction of the nerve elements, there may be little or no increase of nuclei in the commissures. The same is true in regard to sclerosis where the white substance close to the commissures is affected. On the other hand, the nuclei may be greatly multiplied, the central

canal entirely occluded, and yet no characteristic symptoms be recognized. It would seem that some other influence than inflammation is required to originate such changes. What that influence is, and what are the symptoms, if any, attending this change, we have yet to learn.

Michaud describes the mode of origin of the masses of nuclei. They are arranged in small groups or in lines, like the beads of a rosary (Fig. 23), which have a transverse direction. He says that the obliteration of the central canal is unimportant: more significance belongs to the multiplication of nuclei in the gray commissure at a distance from the central canal. He thinks there is no cell membrane enclosing these nuclei. I have not seen a cell membrane, but on teasing apart the nuclei they are seen to be surrounded by a small amount of granular protoplasm.

GRANULAR CORPUSCLES.

Granular corpuscles or granular cells are found in the spinal cord in various affections: there are almost as many opinions in regard to their origin and significance as there are authors. In the last few years this has been debatable ground in the pathology of the spinal cord. Meyer, Roger and Damaschino, Virchow, Jastrowitz, Huguenin, Hayem, Westphal, Adler, Leyden, Obermeier, Moxon, Simon, Eisenlohr, have all discussed the origin and significance of these bodies, some taking one view and some another: their origin has been referred to the nuclei of the blood vessels or the adventitia, to the connective tissue independently of the vessels, to Deiters' cells, to the cells or nuclei of the neuroglia, or to the nerve elements.

The presence of a few granular corpuscles seems to have no pathological significance. Jastrowitz (Arch. f. Psych. u. Nervenkr., II, III) finds them as normal products in both the brain and spinal cord of the fœtus; with the full development of the nervous centres they gradually disappear. Leyden says they are no longer to be considered as characteristic of inflammation. Mayer comes to the conclusion that general weakness of the system and emaciation is a cause of the fatty degeneration of the spinal

vessels, and says that the granular cells may just as well be the residuum of general disturbance of nutrition of the organism as of a fixed local inflammation. He, however, recognizes that these bodies may exert an injurious influence upon the nervous tissue if they are present in excessive numbers.

In all these discussions the question seems to have had reference to the presence of a few granular corpuscles; that they do not necessarily prove the presence of inflammation. If these bodies are found in large numbers crowded together and obscuring the view of other parts it would not be denied that they are an important pathological product, whether it is a process of degeneration or a chronic inflammation which has given rise to the product.

It seems, then, that granular corpuscles may arise from degeneration or fatty change of the nuclei of vessels, or of the nuclei and cells of the neuroglia. Some authors think that the myeline or the axis cylinder of nerve fibres may also give rise to them. That they may arise from the nuclei of vessels is easily shown by teasing or brushing out the vessels so as to free them from surrounding tissues. It is generally admitted that they may arise from the nuclei and cells of the neuroglia.

Observers differ in opinion as to whether granular corpuscles are formed from the medullary sheath and axis cylinder of the nerve fibres. They probably do originate from the medullary sheath. I have never seen any appearance indicating that they arose from the axis cylinders.

Not much can be added to what has been said by others as to the significance of the presence of these bodies. Leyden (Berl. kl. Wochensch., No. 9, 1878) said, at a meeting of the Berlin Medical Society, that it is always difficult to understand how the granular cells arise: he considers the process is an acute or sub-acute inflammation, which may pass into sclerosis. Westphal, at the same meeting, said in reply to Leyden, that he considered the granular cells to belong to a degenerative process, which is certainly not acute, but chronic.

It is scarcely correct to say that the inflammation may pass

into sclerosis. The formation of these bodies is one of the changes found in every case of sclerosis, and the fact that they thus occur in large numbers, while the cellular elements are also found in large numbers, is one proof that they probably arise from the nerve fibres.

After hardening by the use of both bichromate of potassa and alcohol, a crystalline change sometimes takes place in the granular corpuscles, which causes them to act on polarized light.

These bodies are much less numerous in the gray substance than in the white.

AMYLOID BODIES.

Amyloid bodies are found in both the gray and white substances, grouped about vessels and in the cortical layer. They occur in numerous pathological conditions, sometimes in large numbers. Their mode of development, excepting that probably they do not arise from the nerve fibres, but from the nuclei of the vessels or of the connective tissue, and their significance, are not well known. They cannot exert much influence upon the nerve tissue by pressure.

VESSELS.

The vessels are generally altered in every pathological process. One of the most common changes in chronic cases is a thickening of the walls. This thickening is usually confined to the external coat. Only occasionally is the muscular layer affected. At times there is an increase of the nuclei, and sometimes a great distortion of the vessel, because of the irregular distribution of the changes.

The nuclei of the perivascular sheath may be greatly multiplied, so as to obscure the vessel. Sometimes the structures around the vessels are separated from it by a vacant space: either the vessel has collapsed, or the hardening has caused the surrounding tissues to shrink away from the vessel.

There may be fatty degeneration of the elements of the vessel: the nuclei may undergo this change and give rise to granular cells, or the other elements may suffer and drops of

fat may be scattered over the walls of the vessel (Fig. 25), or the muscular layer may undergo a fatty degeneration.

In acute processes there is less likely to be a thickening of the walls: multiplication of nuclei is more frequent, and an exudation around the vessels is not uncommon. This exudation has been frequently described, and from some observers has received the name colloid. It is strongly tinted by carmine, seems to force its way between the nerve fibres, and probably contributes to their destruction. This exudation is very different from that found in certain chronic changes, which is confined within the sheaths of the vessels and is not tinted by carmine. This colloid exudation is found most frequently in and near the commissures.

In acute or sub-acute processes, the blood-vessels may be greatly dilated, and, perhaps, increased in number, or the smallest vessels become more prominent. Their walls then are usually not thickened. Yellow granules may be found around the vessels, and white blood corpuscles or leucocytes may be found outside the vessels.

CAVITIES.

The cavities found in the spinal cord may originate in two ways: either the central canal is dilated, though this seldom occurs, or the cavity is formed *de novo*, at the expense of the gray or white substance just posterior to the central canal. Th. Simon (Arch. f. Psych. u. Nervenkr., v., 1874, p. 120) has given a very full review of all the cases previously reported. Simple dilatation of the central canal was found in only very few cases. More commonly a compressed central canal could be seen in front of the cavity.

He says the walls of the cavities are formed by a tissue composed of fine fibres, in which are embedded numerous round cells, which contain very large nuclei, and only a small amount of protoplasm. Two of his cases, he thinks, are specimens of tumor, with a central cavity formed by softening and disintegration of the proper tissue of the tumor.

Leyden considers that in such cases there is not only an inflammatory process, but that there is a true, new formation, of the nature of a *glio*-sarcoma.

Wladimir Roth (Arch. de Physiol., 1878, p. 612) also looks on the new-formed tissue as a glioma. Friedrich Schultze (Arch. f. Psych. u. Nervenkr., VIII., 1878, p. 367) reports a somewhat similar case, in which the new growth had the characteristics of a round-cell sarcoma.

August Eickholt (Arch. f. Psych. u. Nervenkr., X., 1880, p. 613) reports a case in which the central canal was itself enlarged, and its walls were very much thickened with an increase of cells and fibres, closely resembling the glioma.

Adolf Strümpell (Arch. f. Psych. u. Nervenkr., X., 1880, p. 695) reports a case in which there was a large central cavity, .07 ctm. long, .03 ctm. wide, which was simply the dilated central canal.

It has been my fortune to examine a cord containing a large cavity confined chiefly to the posterior columns. The central canal, still of considerable size, but rather distorted, lay anteriorly, separated from the cavity by the gray commissure. The walls of the cavity had collapsed when its contents had escaped, and were partially covered with the granular remains of the coagulated fluid. The walls were formed of fibres and cells. The fibres were broad and coarse, several times thicker than the normal neuroglia fibres. Near the cavity these fibres were interspersed with cells, forming a narrow and firm lining membrane; externally the fibres were more loosely woven together, with fewer cells and many granular corpuscles. The cells were either round, containing little protoplasm, with a large round nucleus, or they were fusiform, with an oval, sometimes round, nucleus. These fusiform cells gave rise to fibres exactly resembling the fibres interwoven among the cells. Both cells and fibres were deeply tinted by carmine.

CONCLUSION.

It will be interesting to briefly notice the manner in which these changes are combined to produce various forms of disease. Some authors consider that all inflammations of the spinal cord are interstitial. (Fox, C. Lange.) This is not strictly true.

ACUTE MYELITIS.

Acute inflammation, ending in softening, generally affects all the elements of the cord, and its structure is soon so changed that it is impossible to recognize the two forms of inflammation. The final stage is softening, the cord breaking down into a fluid or semi-fluid pulp. The changes in the nerve fibres which lead to softening have been already considered (page 15). In the neuroglia there is first a swelling of the fibres and of the cellular elements, then fatty degeneration of the latter: the fibres become brittle, granular corpuscles are formed, and these, with the other elements, are finally resolved into a granular, fatty, semi-fluid débris. At the commencement of this process the nerve fibres and cells suffer in their nutrition, and are finally destroyed; but the earliest change is in the neuroglia.

The blood-vessels may be first affected: then the changes in the nervous tissues and neuroglia are secondary, and partake more of the character of necrosis than of inflammation. It may be very difficult to decide which change is primary or which secondary. Occasionally the diseased blood-vessels rupture. Sometimes pus is found mingled with other products of inflammatory softening. Sometimes pus is found in the larger septa, having penetrated from the periphery in case of meningo-myelitis.

In most cases of acute softening, the interstitial tissue is probably first affected, the nerve fibres in these cases being destroyed secondarily: yet the destruction of these nerve fibres depends so intimately upon the disease of the neuroglia, and follows that so closely it is almost synchronous therewith.

In acute parenchymatous myelitis the progress is less rapid than in acute softening from interstitial myelitis; the disease invades successively one zone after another, advancing more rapidly longitudinally than transversely. In these cases the changes are such as to show an irritation or inflammation of the proper nervous elements: the nerve fibres are enlarged, axis cylinders hypertrophied; the fibres break up into fat and granular corpuscles, and

the nervous elements disappear before the neuroglia is seriously affected. In such a case the cord retains its consistency, is not much if at all softened; it may be rather more yellow than normal on section.

It seems now to be generally admitted that in infantile paralysis and allied affections in the adult the nerve cells are primarily affected, other changes being secondary. It is interesting in connection with the affection of nerve cells alone, that Eichorst (Virch. Arch., LXIV., 1875, p. 425) states that in fœtal life the anterior cornua are very rich in blood-vessels, and these are so distributed that each nerve cell is surrounded by a net-work of vessels.

In Gombault's case of paralysis in the adult (Arch. de Physiol., v., 1873, p. 80) the nerve cells alone were affected, the white substance was healthy, there was no increase of nuclei in the gray substance, and the vessels were healthy.

CHRONIC MYELITIS.

CHRONIC INTERSTITIAL MYELITIS.

In this form of myelitis, which is usually called sclerosis, the nuclei are increased in number, the spider cells become larger, are more easily recognized, and probably are more numerous; the walls of the blood-vessels are thickened, the nuclei in the perivascular sheath or the adventitia may be multiplied; the fibres of the interstitial tissue become more numerous. Either from compression or from direct interference of their nutritive supply the medullary sheath of the nerve fibre disappears: the axis cylinder may become enlarged but not to such an extent as is seen in acute myelitis. The medullary sheath may simply disappear, or it may break up into bodies resembling granular corpuscles. It has been said that the swelling of the axis cylinder is the first stage of its disintegration and that it then changes into granular corpuscles: but it seems more probable that the axis cylinder simply suffers atrophy and finally disappears. Many, however, persist indefinitely after their medullary sheaths are gone. After hardening and preparing with turpentine, etc., the

transverse section of the cord often shows small cavities (Fig. 24, *o*) not filled with fibres nor cells, not containing axis cylinders, either empty or filled with an indistinct granular mass, which seems to be the remains of granular corpuscles. In some cases these cavities lie near together, but are almost always separated by a narrow septum of interstitial tissue: sometimes they are scattered singly over the field. Their origin is rather uncertain; apparently they have been filled with granular corpuscles, the product of disintegration of tissue.

As the fibrous septa are distributed variously in the different regions of the cord, the manner in which the enlarged, thickened septa seem to force their way between and among the nerve fibres, in the earlier stages of the process, must vary in different regions. In the anterior columns the septa run a course from the periphery to the centre, nearly parallel one with another, anastomosing with each other by short and thin processes: in these columns the inflammatory thickening of these processes gives to the sclerosis in its first stage a radiating appearance. In the lateral columns the septa are less regular, they interlace in a more intricate net-work, which is rather coarse near the central gray substance. In these columns sclerosis, in its earlier stage, gives a coarsely reticulated appearance to the altered tissues. In the posterior columns the septa are more finely reticulated in the columns of Goll, rather coarsely reticulated in the external radical columns. Sclerosis follows this arrangement and in the central parts of the posterior columns the change is more irregularly distributed in the first stage than in any other part. The inflammatory proliferation of fibres can sometimes be seen to arise from several separate points, where are the enlarged and prominent spider cells.

These variations in structure, as explaining the progress of the disease in different regions, will also explain the grouping of the nerve fibres which are comparatively intact. In the anterior columns they are in chains or rows of three or four or more, radiating towards the periphery, in the lateral columns in smaller groups: but everywhere it is evident that

the cause of such aggregation is the normal distribution of the septa.

In the formed chronic interstitial myelitis, sclerosis, there is seen a tissue composed of extremely fine fibres interwoven closely around nuclei, spider cells, axis cylinders, and the cavities mentioned above, which may in freshly prepared specimens, and especially with a low power, seem to be merely a mass of granular substance enclosing vessels with thickened walls.

After a while, if the patient lives, the granular corpuscles may be nearly or quite absorbed, so that they may not be as numerous in one section as in another, or as in another case.

Corpora amylacea are found scattered over the diseased tracts, and in their vicinity, in varying numbers.

Chronic interstitial myelitis may affect the gray substance as well as the white. Then there is an increase of nuclei, the spider cells become more prominent, and probably are also increased in number. As in the white substance the axis-cylinders persist, and the neuroglia cells become more prominent, and probably are also increased in number; the nerve cells seem at first to be but little affected: they retain their form and size, excepting, perhaps, towards the very last stage of the disease.

CHRONIC PARENCHYMATOUS MYELITIS.

In this form of myelitis, which is familiar to us as the lesion found in locomotor ataxia, and perhaps in symmetrical lateral sclerosis, the elements of the interstitial tissue are increased, but less than in interstitial myelitis. The nerve fibres which persist in a nearly healthy condition are found not only near the borders of the disease, but scattered singly or in small groups over the affected spot, unless the change is far advanced. The persisting fibres are generally smaller than normal, the axis cylinders most frequently disappear with the medullary sheaths. Charcot says they are wanting, but I think that I have seen them after the medullary sheath has disappeared: they are, however, soon absorbed. The nerve fibres being primarily and most seriously affected, it is reasonable to follow Charcot in calling this parenchymatous

myelitis. It is interesting to notice in this connection that true muscular atrophy, depending upon lesion of the cells of the anterior cornua, a parenchymatous change, is found much more frequently in these cases than in the so-called sclerosis, the chronic interstitial myelitis.

Leyden (Arch. f. Psych. u. Nervenk., x., 1880, p. 641) says, in regard to a case of amyatrophic bulbar-paralysis and symmetrical lateral sclerosis, "hence we come to the conviction that we have to do with a chronic parenchymatous degenerative process advancing in the nervous elements in the course of those fibres having the same functions, in which the changes in the neuroglia play a subordinate part, and are developed only secondarily."

Schultz (Deut. Arch. f. kl. Med., xxiii., 1879, p. 343) thinks that the existence of a primary sclerosis of the lateral columns is not yet established.

In sclerosis the boundary between diseased and healthy tissue is not a well-defined line; the trabeculae of connective tissue thicken and gradually push their way in between the nerve fibres: in these thickened septa the nuclei and cells are increased: the transition is gradual, but even in small spots of sclerosis and in places where it is seemingly of recent formation, there is a limit beyond which there are no nerve fibres, only axis cylinders. In ataxia the disease shows no marked boundary line, the healthy and diseased portions shade off into each other, but in rather a different way. The nerve fibres diminish in number, and the interstitial substance increases, but the latter does not push out into the healthy districts, widening and thickening as in sclerosis. Groups of nerve fibres vanish, others remain: where the disease is more advanced those groups are composed of a smaller number, or there may be only single nerve fibres scattered over the field. Not until the disease is far advanced, and has invaded nearly the whole of the posterior columns, is there seen the total loss of medullated nerve fibres which is observed at a comparatively early period in sclerosis. That this difference does not depend upon the anatomical structure of the posterior columns is shown by the fact that when sclerosis passes over from the lateral to the posterior columns, the degeneration of

the nerve fibres follows the same rule as when the sclerosis is confined to the antero-lateral columns.

Again, in sclerosis the axis cylinder persists for a long time. In ataxia the axis cylinder disappears much earlier in the disease.

In ataxia the blood-vessels are occasionally covered with nuclei, but to a less general extent than is found in sclerosis. In ataxia there is more generally hyaline thickening of the walls of the vessels, without increase of nuclei.

As to the nature of this process, Leyden (Klinik., IX., p. 330) says, that it seems most probable that atrophy of the nerve fibres is the starting-point of the process; "that, according to Charcot's expression, it is a parenchymatous sclerosis."

Adamkiewicz (Arch. f. Psych. u. Nervenk., X., 1880, p. 772) reports a case in which were some of the symptoms of ataxia: the lesions he thought were primarily interstitial. From this he concludes "that tabes is a chronic degeneration of the connective tissue." Yet the changes he describes are quite different from those usually seen in locomotor ataxia, — so different that no conclusion can be drawn from his case justifying the above view.

Strümpel (Arch. f. Psych. u. Nervenk., XI., 1880, p. 27) carefully analyzes three cases, two of lateral sclerosis (degeneration of pyramidal columns), also degeneration of the posterior columns, and one a case of more decidedly tabetic character. He concludes that these cases must be considered as cases of parenchymatous degeneration. His paper is well worth reading.

Some authors have considered that granular corpuscles are rare or are absent in ataxia. They are rare in the older parts, but present and numerous in the more recent parts, of the disease.

The distribution of chronic interstitial myelitis in patches, scattered here and there irregularly through the cord, is explained by the fact, that in the interstitial tissues new centres of origin give rise to local extension of the disease: as the connective tissue extends irregularly without bearing any direct relation to the development or functional grouping of

the nerve fibres, the spots of disease extend irregularly, and hence may invade any or all the white columns, and encroach upon the gray substance.

In chronic parenchymatous myelitis, the nerve fibres being at first chiefly affected, the disease extends in the direction of the nerve fibres, and affects one and the same bundle of fibres over a relatively long tract: thus, in one case, the external radical columns were affected in the lumbar, dorsal, and cervical regions: in only a portion of this extent were the columns of Goll involved.

In progressive muscular atrophy the nerve cells are chiefly or exclusively affected, the interstitial tissue escaping. The nerve cells pass through the changes already described. In many of these cases there is, however, finally, some change of neuroglia.

There may be, then: —

1. *Acute interstitial myelitis*, with swelling of the fibres, nuclei, and cells of the neuroglia, with destruction of nerve fibres and nerve cells, leading to softening.

2. *Acute parenchymatous myelitis*, where the nerve fibres in the white substance are primarily or chiefly affected, myeline and axis cylinders both disappearing, but the interstitial tissue remaining, seemingly not much changed: also cases in which the nerve cells are chiefly affected, especially those of the anterior cornua, the nuclei and cells of the neuroglia being almost entirely exempt from change, as in infantile paralysis and allied affections.

3. *Chronic interstitial myelitis*, affecting the neuroglia, fibres, nuclei, and cells in both white and gray substance, the nerve fibres and cells being affected only secondarily, as in sclerosis.

4. *Chronic parenchymatous myelitis* — in the white columns only locomotor ataxia, or lesion of the posterior columns (and secondary ascending and descending degeneration possibly), is as yet well known: lateral sclerosis probably belongs to this variety. In the gray substance the cells are affected as in progressive muscular atrophy.

There is as much reason to thus subdivide myelitis as there is to divide nephritis into the interstitial and parenchymatous forms.

EXPLANATION OF PLATES.

Plate I. — Healthy Structure.[1]

Figure 1. Portion of anterior column, near cortex, healthy. *a*, cortical layer. *b*, trabecula entering the cord, dividing. *d*, nerve fibres, axis cylinders in centre. Small nuclei, *c*, are seen, especially in the trabecula.

Fig. 2. Spider cells teased out, from posterior column near the commissure.

Fig. 3. Longitudinal section, showing nerve fibres, some running at an angle to the others; neuroglia fibres running parallel with nerve fibres, or a few transversely; nuclei scattered over the section. *a'*, medullary sheath showing around axis.

Fig. 4. Transverse section through posterior column near commissure, dorsal region, showing the grouping of nerve fibres and spider cells. *c*, spider cell with two fibres from nucleolus. *v*, a small vessel.

Fig. 5. Nerve cell from anterior cornu, healthy, fibres passing from a subnucleolus through the nucleolus and nucleus into the body of the cell (10 immersion).

Fig. 6. Nerve cell from posterior cornu, healthy, shorter fibres from nucleus.

Figs. 7, 8. Nerve cells, healthy, from Clarke's columns.

Figs. 9, 10. Small fusiform cell from posterior cornu.

Fig. 11. Epithelium lining central canal from fœtus at term. Fibres running into commissure are seen.

Fig. 12. A small vessel with perivascular sheath.

Plate II., III. — Pathological.

Fig. 13. First stage of chronic interstitial myelitis (sclerosis). *a a*, axis cylinders without myeline. *n*, a group of nuclei of the neuroglia, which are granular. These nuclei are much more numerous than in health. *b*, *c*, nerve fibres with myeline coagulated around the axis cylinders, without concentric markings. *f*, nerve fibre nearly or quite healthy. *e*, fibrous septum.

Fig. 14. Nuclei from sclerosis; *a*, with protoplasm, but no cell wall; *b*, without protoplasm.

Fig. 15. A group of spider cells from a case of sclerosis. The granular matter and fibrous tissue is omitted; *c*, axis cylinders; *a*, large spider cell with numerous processes and two nuclei; *b*, spider cell without nucleus.

Fig. 16. Fusiform neuroglia cells with processes radiating from both ends. One has two distinct nucleoli (10 immersion).

Fig. 17. Enlarged, or swollen spider cell from gray substance in a case of syphilitic disease.

Fig. 18. Spider cell with a group of five nuclei.

Fig. 19. Nuclei and axis cylinders in a case of advanced sclerosis; *a*, small spider cells as if partially atrophied.

Fig. 20. Sclerosis; *v*, vessel with perivascular sheath distended with coagulated material and connected with a spider cell, *s*, by a process which seemed to have a double wall; this cell has another process which divides, sending one branch towards the vessel; *a*, axis cylinders; *n*, nuclei.

Fig. 21. Spider cell with two processes, *a*, *a*, which split up into fibrilæ (magnified 900 diam.).

Fig. 22. Nerve cells in different stages of atrophy; *a*, *b*, *c*, *d*, from one specimen; *e*, *f*, from another.

Fig. 23. Commissure a short distance laterally from the central canal; multiplication of nuclei in its first stage, seen best at *b*; *a*, fusiform cells connected with fibres; one at *a'*, has two nucleoli.

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[1] Figs. 5, 16, 21, are more highly magnified, 25, 26, 27, 28, less highly, than the others.

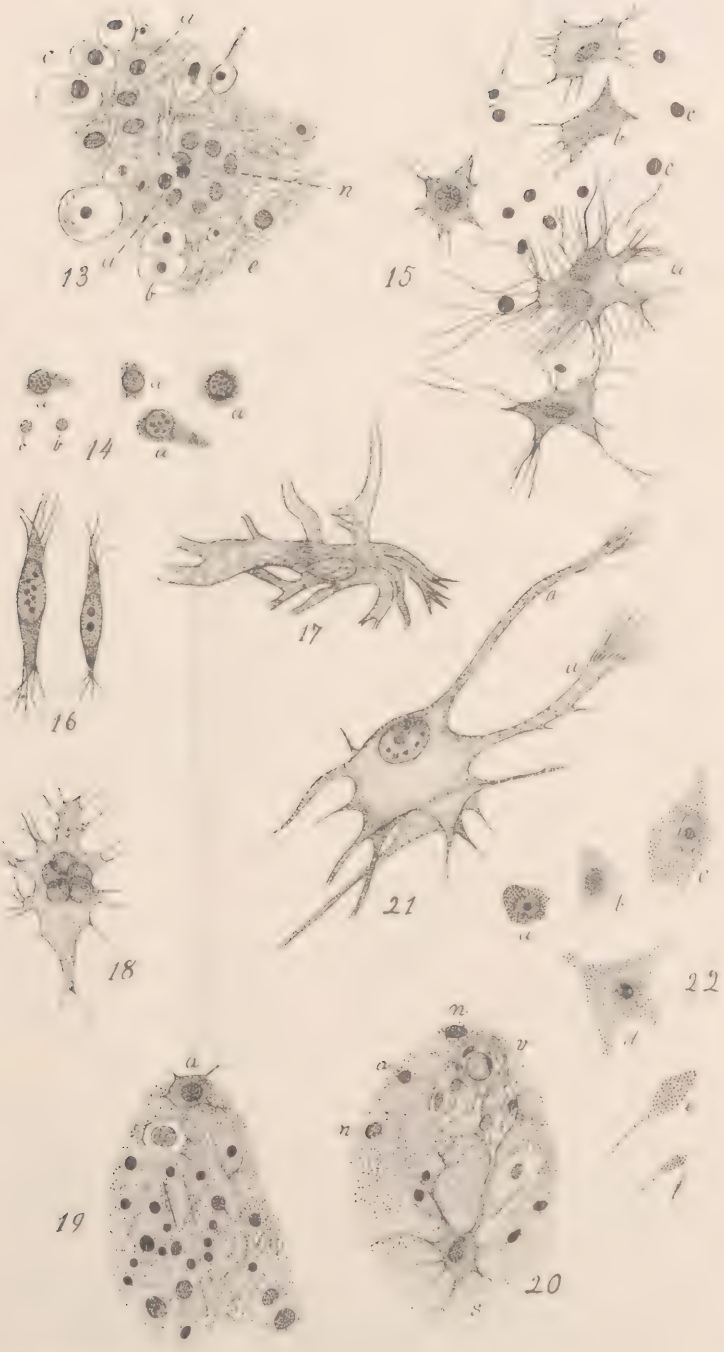
FIG. 24. Sclerosis; *c*, *c*, *c'*, spider cells; *c'*, is connected with the vessel, *v*. There are many small cavities, as at *o*, which, in the fresh state, are filled with granular corpuscles, and as prepared show faint traces of coagulated contents.

FIG. 25. A vessel rather thickly covered with fat globules, from a case of cerebro-spinal meningitis.

FIG. 26. Vessels in sclerosis, transverse section, showing exudation into the perivascular sheath, a moderate thickening of the walls in two of the vessels. A very low power.

FIG. 27. A, transverse section of cord in acute myelitis, showing enlargement of many axis cylinders. B, section of healthy nerve fibres from the same cord at another level. Both these are from the posterior columns. Low power.

FIG. 28. Longitudinal section in acute myelitis, showing hypertrophied axis cylinders. At *a*, showing how they are grouped, overlapping each other; *a'*, *a'* axis cylinders, hypertrophied, containing vacuoles; *v*, vessel. Low power.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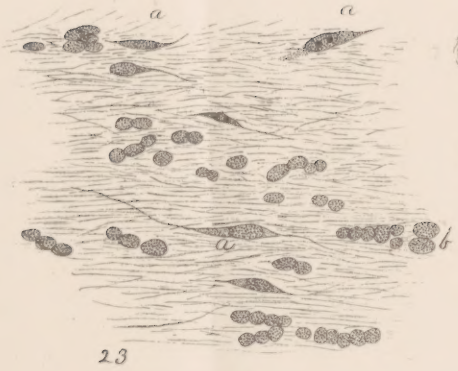

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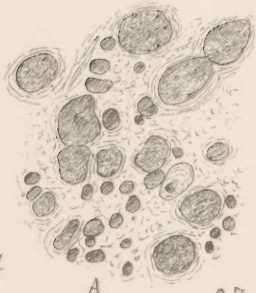

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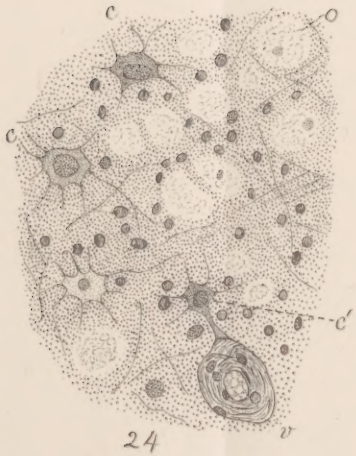

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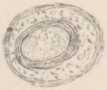

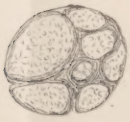

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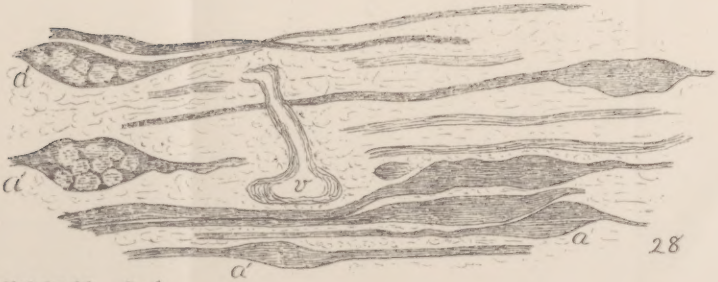

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S. G. Webber Del.